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16. ABSTRACT

In a 22.7-m^3 flow mode smog chamber, 5.4 ppm proylene was allowed to react with 0.9 ppm ozone either in the presence or absence of 0.2 ppm nitrogen dioxide. The steady-state reactant and product distribution was then tested for total mutagenic activity by exposing *Salmonella typhimurium* strain TA100 to the gas-phase chamber effluent. The total product dosage in the test plates was varied by exposing them for 0, 5, 10, 15, and 20 h. *Salmonella typhimurium* survivor levels were obtained at each length of exposure. The number of revertants/plate increased at a rate of ~4-5 per hour, while the survivor level decreased throughout the exposure. Most of the total mutagenic activity can be accounted for by the presence of formaldehyde. This work clearly demonstrates that a gas-phase exposure of *Salmonella typhimurium* can be effectively employed as an alternative to the standard plate incorporation test for volatile species and for complex gas-phase mixtures.

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The Mutagenic Activity of the Products of Ozone Reaction with Propylene in the Presence and Absence of Nitrogen Dioxide

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■ This study was performed to determine if propylene reaction with ozone could account for the large mutagenic activity we have observed in irradiated propylene/NO_x mixtures. In a 22.7-m^3 flow mode smog chamber, 5.4 ppm of propylene was allowed to react with 0.9 ppm of ozone either in the presence or in the absence of 0.2 ppm of nitrogen dioxide (at 25 °C in the dark). The steady-state reactant and product distribution was then tested for total mutagenic activity by exposing *Salmonella typhimurium* strain TA100 to the gas-phase chamber effluent. The total product dosage in the test plates was varied by exposing them for 0, 5, 10, 15, and 20 h. *Salmonella typhimurium* survivor levels were obtained at each length of exposure. The number of revertants per plate increased at a rate of ~4–5 per hour, while the survivor level decreased throughout the exposure. Most of the total mutagenic activity can be accounted for by the presence of formaldehyde. The total mutagenic activity observed was, however, much smaller than that observed in the irradiated propylene/NO_x system, for comparable amounts of propylene consumed.

Introduction

There has been increased concern in recent years that the photochemical conversion of gas-phase pollutants can lead to the production of atmospheric mutagens (*1*). We have recently demonstrated that a simulated urban air mass can be tested for total mutagenic activity using an adapted version of the Ames test (*2*), by allowing gas-phase pollutants to dissolve into the test medium during a gas-phase exposure. We have also recently reported that a relatively simple atmospheric hydrocarbon, propylene (C_3H_6), can be photochemically converted into fairly strong gas-phase mutagens (*3*). In the case of C_3H_6, we have found that an exposure of *Salmonella typhimurium* strain TA100 to the products of an irradited C_3H_6/NO_x mixture at a long extent of reaction, in which reactions of ozone (O_3) and NO_3 with C_3H_6 occur, leads to a substantially increased mutagenic activity. At shorter reaction times, where the dominant C_3H_6 reaction path is with OH radicals, moderate mutagenic activity was observed. In addition, we have recently found that C_3H_6 reaction with NO_3 could not account for a significant fraction of the total response at long extents of reaction (*3*). The major reaction products, i.e., ozone, formaldehyde, acetaldehyde, nitric acid, hydrogen peroxide, and peroxyacetyl nitrate, were also shown to contribute only a small fraction of the total response. We, therefore, have turned our attention toward reactions of O_3 with C_3H_6, with the idea that some unidentified ozonolysis product might be found to be a strong mutagen.

To assess the extent that the products of the C_3H_6/O_3 reaction are mutagens, we mixed ~5.4 ppm of C_3H_6 and 0.9 ppm of O_3 in a continuously stirred tank reactor (in the dark) at a reactor residence time of $\tau = 2.7$ h (where τ = chamber volume/total flow rate). The steady-state distribution of reactants and products was allowed to flow through exposure chambers loaded with Petri plates containing *S. typhimurium* strain TA100. The bacteria were then exposed to the effluent for 0, 5, 10, 15, and 20 h by covering the plates at these times. A possibility that we also wished to address was whether or not the intermediate free radicals present from the ozonolysis reactions could react with NO_2 to yield mutagenic products. We have

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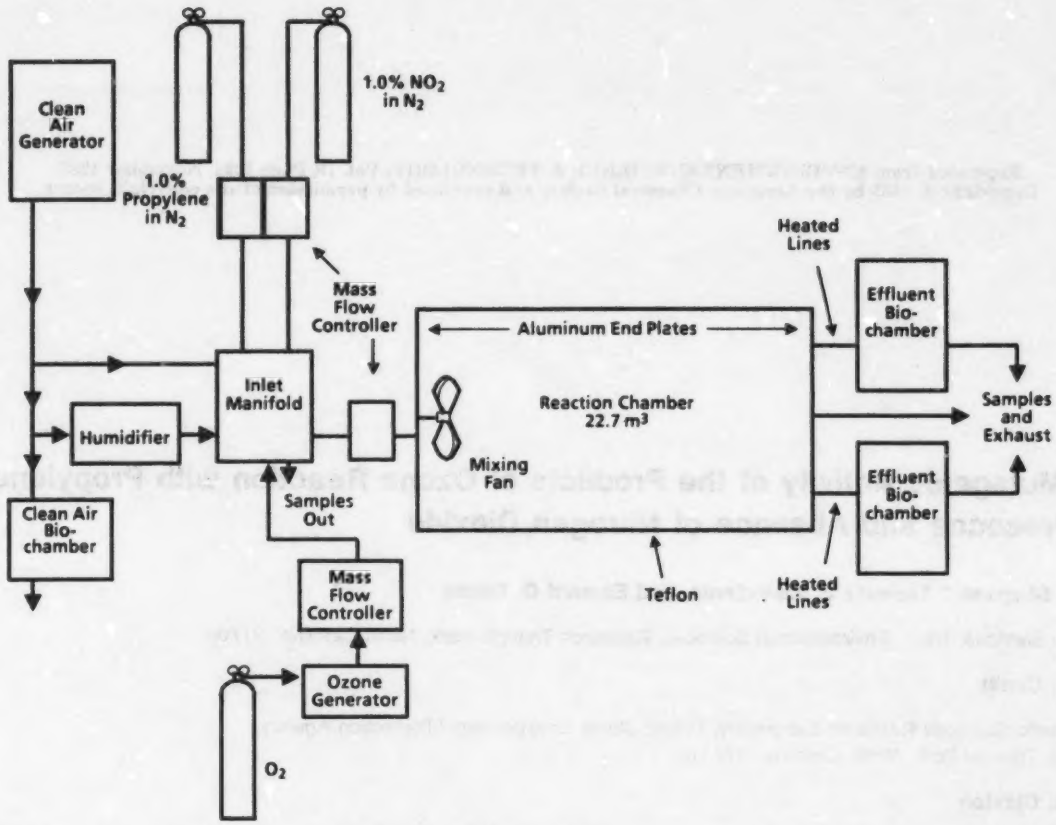

Figure 1. Schematic diagram of the reaction chamber and exposure apparatus.

shown that some organic nitrates and peroxy nitrates (such as PAN) are mutagenic (*3*).

In this paper, we present the results of the bioassays conducted with the products of the O_3 reaction with C_3H_6 in the presence and absence of NO_2. It is shown that most of the response can be accounted for by the presence of formaldehyde (HCHO), which is a weak mutagen. We will also discuss the extent to which the O_3 reaction can account for the observed mutagenic activity in the irradiated C_3H_6/NO_x mixtures. The technique employed is shown to be an effective means of obtaining dose–response curves for volatile compounds.

Experimental Section

These experiments were conducted in a 22.7-m^3 Teflon smog chamber, at 25 °C. (A schematic diagram of the experimental apparatus is presented in Figure 1.) The reactant gases were metered through mass flow controllers into a 150-L stainless steel input manifold, where they were mixed and diluted to their final concentrations. The total input flow was controlled with a 150 L/min mass flow controller. Ozone was produced with a Welsbach Model T-408 O_3 generator supplied with zero grade O_2 (MG Scientific). The NO_2 and C_3H_6 were obtained from MG Scientific as 1% mixtures in N_2. Clean air was provided by an AADCO clean air generator supplied with compressed air. The reaction chamber air did not contain added humidity, although there was some water present due to diffusion through the bag.

During these experiments, the following reactant and product concentrations were periodically measured: C_3H_6, O_3, NO_x, HCHO, peroxyacetyl nitrate (PAN), acetaldehyde (CH_3CHO), propylene glycol dinitrate (PGDN), 2-hydroxypropyl nitrate (2-HPN), and 2-(nitrooxy)propyl alcohol (2-NPA). The analytical procedures employed have been described previously in detail (*2*, *4*).

The exposures were conducted by bringing the reactant (as measured in the inlet manifold) and product concentrations to steady-state values at a reactor residence time of 2.7 h. The reactor effluent was then continuously transferred (at 14 L/min) through heated (~50 °C) Teflon lines (9.5 mm i.d.) to two 190-L exposure chambers. The Teflon lines are heated to maximize transfer efficiency of products to the exposure chambers. Heating of the lines does not significantly increase the exposure chamber temperature (25 °C). The clean air was tested for mutagenic activity (as a control) by passing it through a clean air exposure chamber, as shown in Figure 1.

The biological assay used in this work employs the bacteria *S. typhimurium*, strain TA100. The plates were prepared by adding 0.1 mL of the *S. typhimurium* culture to 3 mL of an agar overlay at 45 °C. This mixture was then poured onto ~45 mL of plate agar in a glass Petri plate. The *S. typhimurium* tester strain TA100 was provided by Dr. Bruce Ames (University of California, Berkeley, CA). Colony counting was done with an Artec 880 automatic colony counter using previously published guidelines (*5*). The test procedures used were those of Ames et al. (*6*), except for the following modifications: (1) glass Petri dishes were used, (2) 45 mL of base agar per plate was used, (3) minimal histidine at the same final total concentration was placed in the bottom agar rather than the top agar, and (4) 3 mL of overlay agar with $\sim 1 \times 10^8$ bacteria was used.

The exposures were conducted by loading each exposure chamber with 35 covered glass Petri dishes containing the *S. typhimurium* test plates and also 10 covered "survivor" plates, diluted by $\sim 10^4$, to which additional histidine was

Table I. Average Reactant and Product Concentrations

species	C_3H_6/O_3, ppm[a]		$C_3H_6/O_3/NO_2$, ppm[c]	
	inlet	effluent	inlet	effluent
C_3H_6	5.41 (0.12)	4.39 (0.10)	5.34 (0.09)	4.39 (0.08)
O_3	0.895 (0.035)	0.076 (0.004)	0.894 (0.032)	0.069 (0.002)
NO_x[b]		(0.229 (0.020))[d]	0.212 (0.012)	0.210 (0.014)
HCHO		0.588 (0.013)		0.599 (0.030)
CH_3CHO		0.355 (0.006)		0.348 (0.007)
PAN		0		0.032 (0.002)
2-HPN[c]		0		10.5 (3.3)
2-NPA[c]		0		2.8 (0.9)
PGDN[c]		0		0.15 (0.02)

[a] Standard deviation in parentheses. [b] NO_2 was added to one of the two effluent biochambers in the C_3H_6/O_3 exposure. [c] Concentrations in ppb. [d] Effluent NO_x biochamber.

added (the approximate 10^4 dilution brings the total plate bacteria concentration to ~500 per plate; therefore, in the absence of toxicity, 500 colonies per plate would form due to the added histidine). Once the exposure chambers were loaded and sealed, the exposure chamber product concentrations were allowed to approach their steady-state values. Once steady state was achieved, 28 of the bioassay test plates and eight of the survivor plates were uncovered, effectively starting the exposures for these plates. The remaining seven test and two survivor plates were covered throughout the exposure, representing a zero dose level. Then, after 5, 10, 15, and 20 h, seven test plates and two survivor plates in each exposure chamber were covered, effectively stopping the exposure at that time. There were, therefore, five different exposure levels in these experiments. At the end of the 20-h exposure period, the covered plates were removed from the exposure chambers and incubated at 37 °C for 48 h, and the final revertant and survivor colonies were counted.

In addition to the test plates, five plates containing deionized water were placed in each chamber. One of the five plates was then exposed for the same time period as the test plates (0, 5, 10, 14, and 20 h). These plates were analyzed at the end of the exposures for nitrite ion (NO_2^-), HCHO, PGDN, 2-HPN, and 2-NPA. Formaldehyde was measured by using the chromotropic acid technique (7), and NO_2^- was measured by ion chromatography as described previously (2). PGDN, 2-HPN, and 2-NPA were analyzed by direct injection of a small sample from the water plates into a gas chromatograph (described in ref 4).

Two experiments were conducted: one without added NO_2 and one with 0.2 ppm of NO_2 introduced into the inlet manifold. We have found that NO_2 solubilizes into water to yield, principally, NO_2^-. Therefore, in the experiment in which NO_2 was not added to the inlet manifold, we added ~0.2 ppm of NO_2 directly to one of the two effluent exposure chambers. In this way, comparison of the results for these two exposure chambers would indicate whether or not reactions of the products with NO_2^- in the test medium to produce mutagens were occurring. In the experiment with added NO_2, the two effluent exposure chambers were identical.

Results

The average reactant (inlet) and product concentrations measured during the C_3H_6/O_3 and $C_3H_6/O_3/NO_2$ 20-h exposures are presented in Table I. The bioassay results are presented in Table II as averages of the seven plates for each of the five exposure periods. In addition, as a measure of toxicity, we report the results of the average of two survivor plates at each exposure level. The survivor plates are a measure of the surviving bacteria concentration

Table II. Bioassay Results (*S. typhimurium* TA100)

chamber	t, h	revertants/plate[a]			
		C_3H_6/O_3		$C_3H_6/O_3/NO_2$	
		test plates	survivors	test plates	survivors
clean air	0	265 (38)	364	181 (19)	774
	5	235 (18)	430	198 (21)	650
	10	264 (55)	451	207 (21)	790
	15	237 (20)	426	208 (31)	720
	20	244 (23)	424	187 (27)	636
effluent "A"	0	206 (24)	411	174 (20)	679
(or	5	243 (49)	358	262 (29)	222
effluent +	10	244 (50)	196	230 (15)	0
NO_2)	15	253 (56)	0	235 (43)	0
	20	290 (71)	0	244 (39)	0
effluent "B"	0	217 (12)	464	184 (30)	423
	5	246 (20)	187	189 (31)	154
	10	264 (16)	46	241 (78)	108
	15	249[b]	51	288 (28)	0
	20	307 (28)	0	302 (50)	0
spontaneous		215 (27)		200 (18)	
NaN_3		559		484	
laboratory survivors			535		785

[a] Standard deviation in parentheses for seven plates at each exposure time. [b] One plate only.

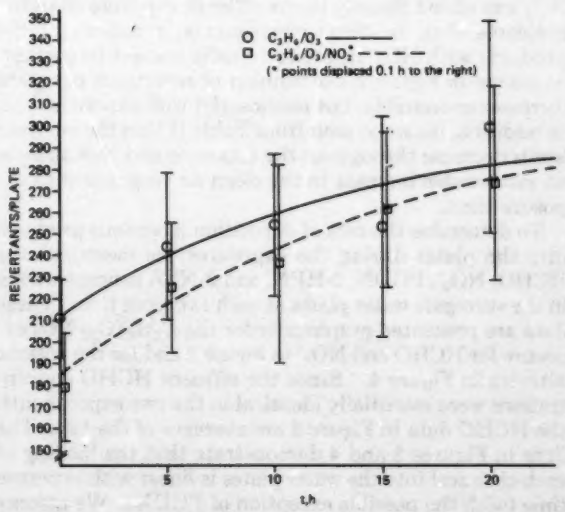

Figure 2. Ames testing data.

(after ~10^4 dilution) at the end of each exposure time. However, since the bacteria concentrations in the survivor plates are different (~10^4 lower) than in the test plates, the survivor data are only an indication of the possibility

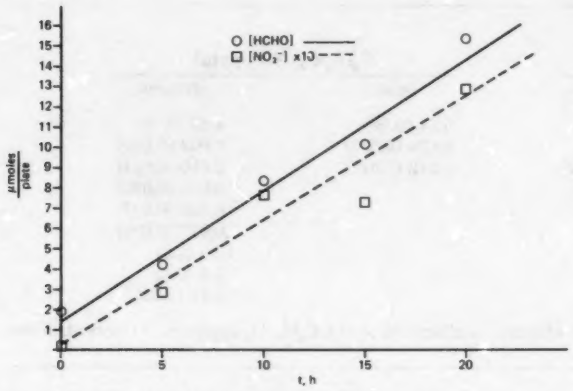

Figure 3. Water plate data, HCHO and NO_2^-.

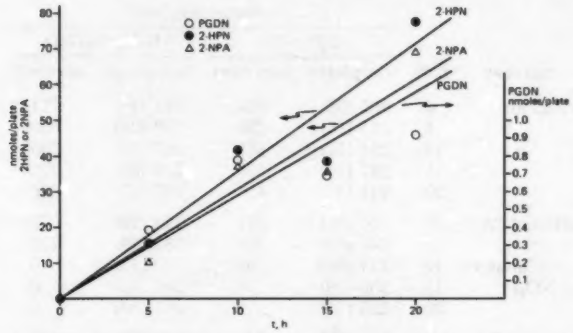

Figure 4. Organic nitrate water plate concentrations.

of toxicity effects, and do not necessarily measure the actual toxicity to the test plate bacteria (*8*). The bioassay data vs. exposure time is presented graphically in Figure 2, along with error bars indicating the ±1 standard deviation uncertainties. Since, from inspection of the data in Table II, it can be seen that there is no significant difference between the results for the two effluent exposure chambers in both experiments, the data in Figure 2 are averages for the two chambers. The experiment in which NO_2 was added directly to one effluent exposure chamber indicates that, in these experiments, reactions of the products with NO_2^- in the test media are not important. As shown in Figure 2, the number of revertants per plate increases measurably (but nonlinearly) with exposure time. In addition, it can be seen from Table II that the survivor levels decrease throughout the exposure and that there is no measurable increase in the clean air response with exposure time.

To determine the rate of deposition of various products into the plates during the exposures, we measured the HCHO, NO_2^-, PGDN, 2-HPN, and 2-NPA concentrations in the surrogate water plates at each exposure time. These data are presented graphically for the $C_3H_6/O_3/NO_2$ exposure for HCHO and NO_2^- in Figure 3 and for the organic nitrates in Figure 4. Since the effluent HCHO concentrations were essentially identical in the two experiments, the HCHO data in Figure 3 are averages of the two. The data in Figures 3 and 4 demonstrate that the loading of each chemical into the water plates is linear with exposure time (with the possible exception of PGDN). We assume this is also the case with the test plates.

Discussion

The C_3H_6 concentration for these experiments was chosen to favor O_3 reaction with C_3H_6 rather than with NO_2, in the case of the $C_3H_6/O_3/NO_2$ exposure. Under

Table III. Reactant and Product Mutagenic Activities, TA100

	species mutagenic activity, revertants/μmol per plate		species mutagenic activity, revertants/μmol per plate
C_3H_6	BD[a]	PAN	35
O_3	BD	2-HPN	3
NO_2	BD	2-NPA	3
HCHO	12	PGDN	BD
CH_3CHO	BD		

[a] ≤2 revertants/μmol

the conditions of this experiment, the initial rates of reactions 1 and 2 are 71 ppb/min and 10 ppb/min, respec-

$$O_3 + C_3H_6 \rightarrow \text{products} \quad (1)$$

$$O_3 + NO_2 \rightarrow NO_3 + O_2 \quad (2)$$

tively, using $k_1 = 1.0 \times 10^{-17}$ cm^3·molecule^{-1}·s^{-1} and $k_2 = 3.7 \times 10^{-17}$ cm^3·molecule^{-1}·s^{-1} (*9*). In this way, the extent of NO_3 reaction with C_3H_6 is minimized. Comparison of the organic nitrate yields with those found in a C_3H_6/N_2O_5 study conducted in our laboratory (*4*) indicates that NO_3 reaction with C_3H_6 occurred to the extent of ~0.05 ppm in the $C_3H_6/O_3/NO_2$ exposure (or only ~5% of the total reacted C_3H_6).

The reaction of O_3 with C_3H_6 can produce various free radicals (e.g., HO_2 and CH_3O_2) (*10*) that may then react with NO_2 to produce peroxy nitrates (e.g., $CH_3O_2NO_2$). From a comparison of the results (Figure 2) for the C_3H_6/O_3 and $C_3H_6/O_3/NO_2$ exposures, it appears that either these are not significantly mutagenic at these concentrations or they are not effectively transported to the exposure chambers.

As shown in Figure 2, the number of revertants per plate increases throughout the exposure at ~4–5 revertants/plate per hour. The "dose–response" curves are, however, nonlinear presumably due to increasing toxicity effects with time, as indicated by the decreasing survivor levels. We can, however, attempt to account for the observed response by comparing the initial slope of the curve with the known mutagenic activities of the products present. We have conducted standard plate incorporation tests and/or gas-phase exposures for each of the reactants and products listed in Table I, and the data from these tests are presented in Table III, as revertants per micromole per plate. From the data in Figure 4 and Table III, it appears that 2-HPN and 2-NPA could account for less than 1 revertant/plate each at 20 h. To determine the contribution to the total response from PAN, we need to estimate the total quantity of PAN that deposits into the plates. In a previous paper, we reported that under these conditions ~15% of the PAN passing through the exposure chambers dissolves into the media (*2*). The PAN concentration deposited into the plates over each exposure period can be estimated from eq I where C_p is the con-

$$C_p = 0.15 C_g F_c T_e (1/N_p) \quad (I)$$

centration in the plates, C_g is the molar gas-phase concentration, F_c is the flow rate through the exposure chambers (840 L/h), T_e is the exposure time, and N_p is the total number of exposed plates. Using this equation, we calculate an initial PAN deposition rate of 4.1 nmol/plate per hour, which leads to a potential contribution from PAN of ~0.14 revertant/plate per hour, or ~3% of the initial reversion rate.

We have found both by standard plate incorporation tests and by gas-phase HCHO exposures that HCHO yields

a response with TA100 of ~12 revertants/μmol per plate. However, the dose–response curves tend to bend over due to toxicity effects (at ~6 μmol/plate) yielding a maximum excess of ~70 revertants/plate. From the slope of the curve for HCHO in Figure 3, it appears that the HCHO can account for ~8 revertants/plate per hour, or 40 excess revertants at 5 h. This is actually greater than the observed reversion rate in the experiment. Our gas-phase exposures with HCHO have indicated essentially no decrease in the survivor level for HCHO doses up to 6 μmol/plate. Therefore, there are probably other species present that are toxic and that lead to a reduced reversion rate. We have previously conducted experiments with ~0.5 ppm of O_3 in the exposure chambers without toxicity effects, indicating that O_3 is not toxic at these levels. It does appear, however, that essentially all of the observed responses in these experiments can be accounted for based on the presence of HCHO.

The reaction of O_3 with C_3H_6 leads to production of several other products (*10*), such as CH_3OOH, H_2O_2, and secondary ozonides. Since these are all probably soluble in the medium, these products either are not transported to the exposure chambers, or are not significantly mutagenic with this strain, at these concentrations.

The principal objective of these experiments was to attempt to determine the extent to which C_3H_6 reactions with O_3 could account for the large response (~600 excess revertants/plate) observed in the irradiated C_3H_6/NO_x system (*3*). Computer simulations of the irradiated C_3H_6/NO_x system under the conditions of that exposure indicated that the C_3H_6 reaction with O_3 had occurred to the extent of ~0.20 ppm of reacted C_3H_6. Since this is only ~20% of the extent of C_3H_6/O_3 reaction in this study, it appears that the products of the C_3H_6/O_3 reaction could not have caused a significant response in the C_3H_6/NO_x irradiations.

Since we have also found (*3*) that the products of the C_3H_6/NO_3 reaction could not have caused a significant response in the C_3H_6/NO_x irradiation, it appears that there may be some very minor product, which is probably photolytically generated, that is a very strong mutagen present in the C_3H_6/NO_x-irradiated system.

It is clear from the data in Figures 2–4 that this technique of gas-phase exposure with variable exposure times presents an effective means for obtaining dose–response curves with volatile species and complex mixtures (provided those species are water soluble). In addition, the use of survivor plates permits us to evaluate the potential for toxicity effects experienced by the bacteria during the exposure.

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